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# Nervous Shock and Disease of the Nervous System as a Cause of Pernicious Anemia.

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# NERVOUS SHOCK AND DISEASE OF THE NERVOUS SYSTEM AS A CAUSE OF PERNICIOUS ANEMIA.

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BY JAMES B. HERRICK, M.D.

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It is the object of this paper to direct attention to nervous shock or disease of the nervous system as a possible, or probable, exciting cause of grave or pernicious anemia. My attention was called to the relations between these conditions by the following case:

The patient, Mrs. M., white, 63 years of age, claimed to have been in good health up to November, 1893. She had rosy cheeks, was plump, weighed 137 pounds, led an active, busy life as a quasi-practitioner of medicine. She scarcely knew what it was to be confined to bed save when her children were born, five of whom she had brought into the world alive and healthy. She had never miscarried and there was no other evidence leading to the suspicion of syphilis. Her father had lived to the age of 87, to die an accidental death. Her mother succumbed to pleurisy at 63. One brother had died from some cardiac trouble and one sister from asthma. Two brothers and one sister were living.

In November, 1893, she fell on the sidewalk, hurting her back and left side. To use her own words: "The hurt extended from the back of the neck down the left side and left leg." She walked home, but from the time of this injury she found herself weaker, both physically and mentally. She was obliged to lie down the greater part of the day, and thought she was becoming paralyzed. Soon the legs began to bloat; and she would, upon sitting up for a short time,





feel dizzy and perhaps faint. The patient became unable to walk for any great distance; had difficulty in picking up small objects such as a pin. The skin became paler; the appetite, notwithstanding, remained good; the bowels were constipated. She was often quite thirsty.

On admission to the Presbyterian Hospital June 30, 1894, her complaint of somewhat vague pain, of weakness of the arms and legs, and mental deterioration, for all which she assigned the injury as the efficient cause, coupled with the fact that there could be discovered no evidence of organic lesion to account for such phenomena, and also that she referred often to a \$2,000 accident insurance claim that she had placed in the hands of a lawyer for collection, all this aroused a suspicion of exaggeration of symptoms as in railway spine, if not of malingering.

The increasing pallor of the skin led later to an investigation of the blood and a more thorough examination of the patient. I condense the results of several examinations.

Physical Examination Nov. 8, 1894.—Skin and mucous membranes of a lemon-yellowish white. Conjunctivæ show light yellowish tinge. Skin is dry, wrinkled, panniculus adiposus scanty. Patient talks rather slowly, as if taking a long time for framing answer and separate words. Speech reminds one of scanning speech of insular sclerosis. She moves hands and legs slowly and somewhat uncertainly; appears weak and unable to walk or stand unsupported; complains of great dizziness on being put in erect posture. Hair is gray, thin and falling out fast; ears and nose negative; tongue pale, flabby, not tremulous, protruded in median line; fetor *ex ore*; frequent spasmodic contraction of lower portion of orbicularis palpebrarum. Right eye beginning cataract; numerous retinal hemorrhages; left eye, retinal field practically the same; in right eye six hemorrhagic areas counted, in left twelve. The existence of retinal hemorrhages was confirmed

by Dr. Alfred Hinde, who kindly examined the eyes for me. External jugular pulsates synchronously with apex-beat as does a vein (probably a perforating branch of the internal mammary) running just to the right of the sternum and parallel with it for its upper one-quarter. The chest is slightly flattened, of moderate length, intercostal spaces rather wide; respiratory movements regular, fairly deep, eighteen to the minute. Apex-beat faintly seen and felt in the fifth interspace just inside the left mamillary line; palpation otherwise negative. Percussion reveals no increased area of cardiac dulness. Pulmonary resonance on the right side in the mamillary line on expiration as low as to the sixth rib, on inspiration over seventh rib. Respiratory sounds are normal save an occasional moist râle; (in the last two days has "taken cold"). Systolic blowing is heard over entire precordia, best at apex; systolic hum also over vessels of neck. Abdomen is flabby, marked with lineæ albicantes; occasional peristaltic movements in region of umbilicus and to right of same. Edge of liver is felt very indistinctly about one inch below costal arch; inguinal glands are palpable but not perceptibly enlarged. No tumor mass or point of tenderness is found on palpation. Liver dulness is heard in median line, one-half way between ensiform cartilage and umbilicus; area of splenic dulness increased in posterior axillary line reaching as high as to the eighth rib; Stomach resonance apparently increased in area. Stomach reaches below umbilicus (gastroptosis); it appears moderately enlarged. Lesser curvature apparently made out between ensiform and umbilicus. After test breakfast (Ewald) no free HCl, no mucus, no remains of previous meal are present; pepsin uncertain; no lactic acid by Uffelmann's test. Lower extremities are moderately edematous; rectum negative, no parasites in stools; pelvic organs negative; bones negative, no tenderness. No evidence of organic or local nervous lesions can be made out. Sensation seems to be perfect, the reflexes normal. There is no paralysis. The



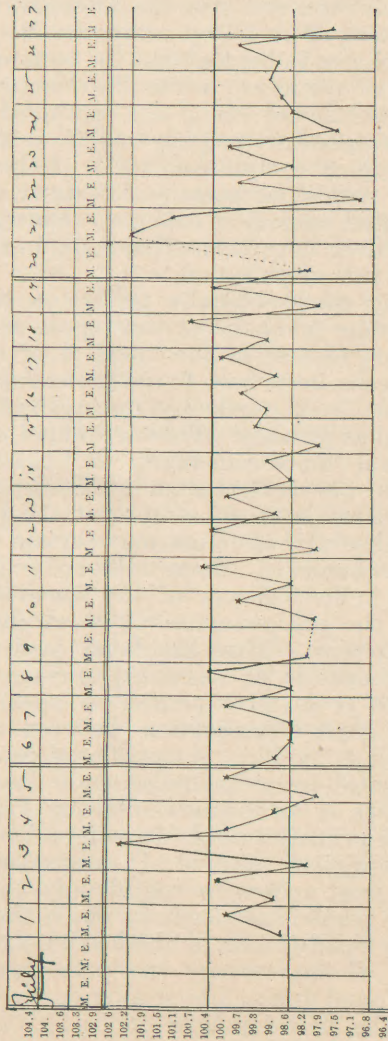
bowels are inclined to constipation. There is no great frequency of urination, no difficulty in urinating, or irritation from urine. Movements of muscles are slow and feeble, not coördinated with certainty. Raises dynamometer to 10 with either hand. Urine is 1012, 30 to 50 ounces; no albumin, no sugar; solids, 660 grains; no formed elements. Blood is pale; marked poikilocytosis, microcytes and macrocytes; few nucleated red; megaloblasts, *i.e.*, the large nucleated reds, few; leucocytes increased relatively; many lymphocytes; number of red globules to cubic millimeter 666,666; hemoglobin (Fleischl) 25 per cent.; no parasites. I desire to acknowledge the kindly aid of the late Dr. D. D. Bishop. Several of the numerous blood examinations were made by him.

I present temperature chart showing continuous irregular fever.

The patient remained in the hospital, though not under my immediate observation, until the middle of the summer of 1895. On increasing doses of arsenic there was a perceptible improvement, both as regards the subjective and objective symptoms. There was less dizziness, more certain movements of the hands and legs, clearer mind, greater strength. The blood count gradually changed so that on March 22, 1895, the hemoglobin was 80 per cent. and the red corpuscles 2,575,000, with no increase in the white. A severe attack of bronchitis or influenza seemed about to carry off the patient, but she rallied from this and finally left the hospital at her own request. I am unable to state facts concerning her subsequent history.

The case was seen by several physicians and all failed to locate any organic cord or brain lesion or any malignant growth.

The intense pallor of the skin, the subjective sensations of dizziness and palpitation, the great mental and bodily weakness, the retinal hemorrhages, the anemic murmurs, the enormous reduction in the number of red corpuscles with such marked varia-



tions in their form and size, the presence, though in small numbers, of nucleated corpuscles, the increased globular richness in hemoglobin, the corpuscles being reduced to 13 per cent., while the hemoglobin was reduced to but 25 per cent., the continued irregular fever, the temperature ranging from 97 to 102 F., gave an almost typical symptom-complex of the so-called progressive pernicious anemia of Biermer, or the primary essential anemia of Addison. Enlargement of spleen and liver have been often noted in these cases as well as venous pulsation.

But many cases clinically perfect as progressive and pernicious in character and apparently due to some primary disease of the blood or of the blood-making organs, have been found postmortem to have some organic lesion overlooked during life that should be regarded as primary. Thus a hidden carcinoma, an intestinal blood-consuming or toxin-producing parasite, as the *anchylostomum duodenale* or the *bothriocephalus latus*, an atrophy of the gastric and intestinal glandular structure may explain an apparently primary anemia. Some authors incline to put these cases in the category of the essential anemias because of the predominance of blood changes and blood symptoms. Thus Eichhorst says that only those deuteropathic or secondary anemiæ are to be regarded as progressive pernicious anemiæ where there is a great disproportion between cause and effect. (Bd. iv, S. 22.) A case, therefore, of atrophy of the stomach or of *anchylostomum* disease where the symptoms of the anemia completely overshadowed those of the primary trouble he might class as progressive pernicious anemia. It seems better, however, with most writers to regard only the cases as progressive pernicious anemia in which the affection is a primary blood disease, in which so far as our present knowledge of the etiology and pathology goes, no change is found during life or after death save in the blood or the hematopoietic organs. That this is usually fatal is well known. Recoveries, however, are



occasionally recorded. The term progressive pernicious had better be discarded, and the term employed first by Addison, primary essential anemia, substituted.

Looking upon this case as one of primary essential anemia, there remains to be noted the interesting connection between the injury with its nervous shock and the anemia. The patient and her friends assert that from a condition of apparent health, she almost immediately following the injury became an invalid, weak, dizzy, easily fainting, with swollen limbs, pale skin, in a word, anemic. Just how nervous shock produces anemia I will not attempt to say. Yet, if through a shock to the nervous system the secretion of the sweat glands, of the gastric glands, or of the kidneys can be checked or increased, if the heart's action can become rapid and irregular, or perhaps cease altogether, if a chorea or an exophthalmic goitre can be roused to activity, it can be assumed that the function of the blood-producing organs may become perverted, and this suddenly, through the influence of a deranged nervous system. I recall a case of cerebral hemorrhage with aphasia and hemiplegia in which there was fatal anemia; also a fatal case of anemia following upon a sunstroke.

I am inclined to rule out atrophy of the glandular tissue of the stomach, largely from the absence of any symptom indicating a previous catarrhal inflammation, though the emaciation different from the well-preserved condition of pernicious anemia, the absence of mucus and of free hydrochloric acid and the enlargement of the stomach might seem to point in that direction.<sup>1</sup> There was certainly not present the small contracted stomach with exuberant development of connective tissue—cirrhosis ventriculi. The dilatation, more apparent than real, because of dislocation of the organ, was probably, as not infrequently occurs, due to relaxation of the atonic stomach walls. I believe

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<sup>1</sup> Some, for example Strümpell, regard the changes in the glands of the stomach as secondary to the condition of anemia. Likewise the changes in the cord and other nervous structures.

there was no hidden carcinoma as the cause of the anemia because of the comparatively sudden onset; the absence of subjective or objective local symptoms; the unusually severe oligocythemia, reduction of erythrocytes to 1,000,000 per cubic millimeter being unusual in the terminal anemia of carcinoma; the marked improvement under arsenic, the improvement lasting for at least six months, and being scarcely reconcilable with a carcinomatous anemia after it had reached a stage indicated by a blood count of only 666,666; the retinal hemorrhage and the absence of cachexia.

Whether or not an organic disease of the brain or cord or of the sympathetic or peripheral nerves was present no one who saw the case would say. The interesting researches of Minnich in this connection are recalled by some of the symptoms.

While nervous shock or disease of the nervous system is not recognized in many of our text-books as an important etiologic factor in pernicious anemia, a number of cases are reported in which this is the assigned cause.

Under this head are not included those cases of nervous diseases as complications or sequelæ of pernicious anemia, such as degenerations of the posterior columns or the other parts of the cord, etc., and which Minnich has recently gone into so fully. (Cf. also Lichtheim, Trechsel, Burr, etc.)

Eichhorst quotes the case of an author who, after a most severe mental strain, succumbed to a fatal anemia.

Curtin, in 1885, under the title "Nervous Shock as a Cause of Pernicious Anemia," reports the case of a woman, 38 years of age, who was unexpectedly brought face to face with the body of her suicided brother with his throat cut. From that time health began to fail, and in four years and two months she was dead of a grave anemia. No blood examination; no autopsy.

2. A young lady was suddenly and brutally informed



of her brother's death. Nervous prostration followed for many months, then pernicious anemia and death.

3. Practice of Dr. Musser: A woman, aged 42, after an attempt at her own murder by her husband, became nervous, excitable, almost insane; gradual failure of health, pronounced anemia; death in three years.

Curtin quotes also the case of a woman becoming profoundly anemic following fright at the house catching on fire. He also quotes from Mackenzie, who cites the case of Sir H. Marsh, where the young lady, who accidentally poisoned her father, was overwhelmed by grief, took to bed and died of anemia. Also the case of a young man who saw a child run over in the street, was greatly shocked, began to grow anemic; no organic lesion postmortem.

Under Sir William Gull, a young man died in Guy's Hospital of extreme anemia that had developed after he had been attacked by a sheep in a field.

Musser in 1885 gave a résumé of thirty-nine cases of pernicious anemia up to that time reported in America, and quotes cases of Curtin, Osler, Pepper and himself, where anemia seemed to be due to nervous shock.

Hutchinson cites cases in which nervous influence seemed to him to be the exciting cause. 1. A case of his own where mental worry over the death of a wife was the cause. 2. Case in consultation where a manufacturer who had worried about business had become anemic, apparently from no other cause than business worry. 3. Four daughters died in one week of smallpox. The father, apparently from the shock and the excessive grief, developed pernicious anemia.

Dr. Brower cites a case of pernicious anemia developing in a previously healthy young woman following a railway accident. Death in six months. He also calls attention to mental worry as an exciting cause of the milder grades of anemia and of chlorosis, and is authority for the statement that in a student wor-

rying and apprehensive about the approaching final examination, the hemoglobin became reduced to twenty per cent.

Schüle, in three cases of pernicious anemia in the insane, seemed to see some genetic relation between the incurable cerebro-spinal lesions and the progressive anemia. He quotes the experiments of Goltz, Heubel and Von Tarchanoff as tending to prove the influence of the nervous system on blood formation, apart from the trophic influence of the spinal cord.

Macphail also describes pernicious anemia as it developed in two insane patients.

Holst believes that through neurasthenia the secretory nerves and the trophic nerves are influenced in such a way that blood deterioration results. He regards the anemia therefore, in many cases, as a result and not the cause of the neurasthenia.

Hale White refers to a patient who fell on the ice. For fourteen days no symptoms, then weakness, numbness in legs and progressive severe anemia; apparent recovery.

Among those who assign to the nervous system, chiefly through what we must, for want of a more accurate term, call nervous shock, a certain rôle in the production of anemia may be mentioned Heiberg, Fabre, Germain Sée, Trousseau.

A number of observers believe there is some causal connection between lesions of the nervous system and pernicious anemia. Thus Saaski believes the gastrointestinal form depends on nerve atrophy in Meissner's and Auerbach's plexuses. These changes he is inclined to look upon as primary and not as secondary to the anemia, though others reverse the order of pathologic change. He examined two cases and forty-eight control cases.

Brigidi examined the body of a woman, aged 53, dead after two years of anemia. The only weighty postmortem finding was an inflammatory and fatty degenerative change in the celiac ganglia. He thinks the influence on the circulation of the diges-



tive tract might explain the poor digestion and consequent anemia.

Pokrowski, autopsy on a case of pernicious anemia, found changes in cerebellum, fourth ventricle and medulla, and was led to conclude that the finding in the central nervous system must have had an influence, if not the sole influence, in producing the anemia.

Banti refers to a group of anemias—anemia ganglionare—in which he believes the primary change is in the sympathetic nervous system.

Little also regards many cases as due to an irritation of the vaso-motor system.

I feel warranted from my study of this case and a perusal of the literature bearing upon this subject, in drawing the conclusion that in some cases of pernicious anemia there is a causal connection between shock or injury to the nervous system and the resulting anemia. Whether such shock acts by interference with the nervous mechanism of the digestive organs, the stomach, intestines, liver, pancreas, the ultimate result being a severe anemia, or whether through altered nervous influence, there is abnormal performance of function on the part of the hematopoietic organs, it is impossible to say. In assigning to nervous shock an influence in the production of anemia it is not necessary to regard it as the sole cause or even the prime cause. Just as in the case of pneumonia we look upon the pneumococcus as the main cause of the disease, but yet regard exposure to cold as an exciting cause that favors the localization or pathogenic action of the specific organism, so in the case of pernicious anemia, the nervous shock may in some way merely favor the action of some otherwise inert microorganism or toxin, that under these altered circumstances produces a profound or even fatal anemia.

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